The genetics and evolution of 'covitality'

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The Genetics and Evolution of Covitality

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The genetics and evolution of covitality: an introduction

That individuals possess distinct personalities has been known for much longer than the history of scientific psychology. Words describing individuals' behavioral, affective, motivational, and cognitive dispositions are found in the world's languages (Allport and Odbert, 1936), and descriptions of personality are replete in literature (McCrae et al., 2013). Take, for instance, Lady Macbeth's description of her husband as being "too full o' the milk of human kindness" (*Macbeth,* Act 1, scene 5, line 17).

However, talking about and scientifically studying personality are different matters. The scientific study of personality comprises multiple theories, each with different, interconnected views of what personality is, how it should be measured, and what aspects of personality should be studied (Funder, 2007). Trait theories of personality are those most closely linked to the study of SWB (Diener et al., 1999) and other characteristics related to psychological well-being. The scientific history of trait theories of personality can be tied back to Francis Galton (1884), who viewed the rich language related to personality

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descriptions as a road to the scientific understanding of "character." Galton's lexical theory served as the bedrock of much personality research up until this day. In the English-speaking world the early history of this research included a study by Allport and Odbert (1936), who collected and categorized personality descriptors in the English language. This early history also included research on the structure of personality by Cattell (1946) and Tupes and Christal (1961), who applied factor analyses to determine how many dimensions are needed to adequately describe human personality traits.

Trait approaches to personality have not gone unchallenged. In 1968 Walter Mischel's book-length review of personality trait research repeatedly showed that traits were not consistent across situations or across time, and typically correlated with behavior at around .3. These findings and others led Mischel (1968) to conclude that situations are the determinants of human behavior and to a decline in research on traits. In response, trait theorists built up evidence refuting the hypotheses of Mischel and other "situationists." In 1988 Kenrick and Funder revisited this debate, reviewing critical tests which refuted claims made by situationists. For example, trait theorists demonstrated that low correlations between personality traits and behaviors were artifacts of the low reliabilities of single traits as measures of personality and single behaviors, and that aggregating across multiple measures led to correlations far exceeding .3 (Kenrick and Funder, 1988). Similarly, research showing that length of acquaintance was associated with the consistency of observer ratings of personality and that observers who knew individuals in different social contexts showed high levels of agreement made it unlikely that inter-rater agreement was produced by cognitive biases or reflected the effects of situations, respectively (Kenrick and Funder, 1988).

The person–situation debate is seen by some as the forge that shaped modern trait approaches to personality (Kenrick and Funder, 1988). That is to say, while many questions raised by Mischel (1968) have been resolved to the satisfaction of nearly all personality

researchers, the field retains characteristics acquired during the debate. For example, ongoing research that examines association between personality and life outcomes, such as health, and success in education, occupation, and marriage (Deary et al., 2010; Roberts et al., 2007; Ozer and Benet-Martínez, 2006), can be seen as demonstrations that personality traits are stable, predict behavior, and do so across situations. Likewise, efforts in behavior genetics that examine the heritability of personality traits tackle whether personality traits are biological entities (Bouchard and Loehlin, 2001).

None of this is to say that there are no remaining debates within personality psychology. Quite the contrary, given that there is still an ongoing debate about the number and nature of the dimensions that characterize human personality (Eysenck and Eysenck, 1964; Goldberg, 1990; Digman, 1990; Costa and McCrae, 1992a; Cattell et al., 1970; Ashton and Lee, 2007).

For this chapter, we will focus on the models known as the Big Five or Five-Factor Model. These models specify that personality traits describe five domains or factors most commonly named *Neuroticism*, *Extraversion*, *Openness to Experience*, *Agreeableness*, and *Conscientiousness*.[3] While comprehensive descriptions of these dimensions can be found elsewhere (Digman, 1990; Costa and McCrae, 1992b; John et al., 2008), it is worth briefly describing them here. *Neuroticism* describes tendencies related to anxiety, emotional instability, negative affect, and vulnerability. *Extraversion* describes the tendency to be sociable and warm, to seek out excitement, and to experience positive emotions. *Openness to*

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[3] Other names for Neuroticism include Emotionality or, when reversed, Emotional Stability. Some alternatives for Extraversion, Openness, and Conscientiousness have included Surgency, Intellect or Culture, and Dependability, respectively.

Experience is related to broad interests, liberal attitudes, aesthetics, and intellectual pursuits. *Agreeableness* describes tendencies to be cooperative, altruistic, kind, and tender-minded. *Conscientiousness* describes the tendency to be goal-directed, self-disciplined, and orderly.

Our decision to focus on the Big Five or Five-Factor Model is pragmatic. The Five-Factor Model is perhaps the most widely used personality model to date (John et al., 2008). This stems from several observations, including the fact that the Five-Factor Model dimensions are found in a range of personality instruments, models, and approaches (see, e.g., Costa and McCrae, 1992b), and have been identified in a wide range of human cultures (McCrae et al., 2005; Schmitt et al., 2007). As such, it is viewed as a comprehensive model of personality that is not culture-bound. In addition, the five factors, as well as their lower-order facets, have been the focus of many twin studies and were found to be heritable (Bouchard and Loehlin, 2001). Finally, the Five-Factor Model and its components have often been studied in the context of SWB (DeNeve and Cooper, 1998; Steel et al., 2008) and other positive psychological constructs. That being said, there is no reason to believe that the associations discussed here are limited to the Five-Factor Model, and they are also likely to be found in the models of Eysenck, Cattell, Ashton, and even in models that have not yet been developed.

What is covitality?

Covitality has been defined as the observation that different positive traits tend to be found together in individuals (Weiss et al., 2002). Covitality is thus the mirror image of comorbidity, which, in the context of psychiatry, refers to the observation that a large proportion of individuals who are diagnosed with one psychiatric disorder are also diagnosed with one or more other disorders (Kessler et al., 2005). In fact, a recent study of the factor structure of psychopathology in the Dunedin cohort showed that a general factor—a so-called

p-factor—best explained the variation among a range of disorders (Caspi et al., 2013). These findings, as well as those showing that individuals who have psychiatric disorders are often in poorer health, exhibit poorer interpersonal functioning, and are at greater risk of early death (De Hert et al., 2011; Harris and Barraclough, 1998), suggest that psychiatric comorbidity extends beyond a single domain of health and functioning.

On the other hand, covitality is displayed by findings that people who score higher in measures of life satisfaction and positive affect, are healthier and live longer, enjoy better interpersonal relationships, and tend to be higher in socioeconomic status (Lyubomirsky et al., 2005; Diener and Chan, 2011). Individuals who report higher SWB also have lower scores on the Hypochondriasis, Depression, Hysteria, Psychopathic Deviance, Paranoia, Psychasthenia, Schizophrenia, Hypomania, and Family Conflict scales of the Minnesota Multiphasic Personality Inventory (MMPI) (Diener and Seligman, 2002). Another positive trait is that of optimism—the tendency to view the future in a positive light. Like people with higher SWB, optimists tend to be psychologically and physically healthier than pessimists (Scheier and Carver, 1992; Rasmussen et al., 2009; see also Chapter 8). Finally, a recent study found that among college students, hedonia, optimism, self-efficacy, hope, and gratitude described a single factor that, in turn, predicted better adjustment and fewer internalizing problems (Jones et al., 2013).

An early set of findings that could be described as covitality came from Lewis Terman's Genetic Study of Genius (1925). Terman's goal was to test the notion, held by many in the United States at the time (and even today), that children with high IQs were sickly, maladjusted, and disadvantaged in other respects. Terman (1925) found the opposite: the study participants, all of whom had IQ scores of at least 135, generally did better in all areas of their lives. They had more prestigious professions, earned considerably more than the national average, were taller, healthier, and enjoyed more successful relationships (Oden,

1968). Recent studies find similar results related to health in samples spanning the normal range of intelligence scores (see Deary et al., 2010 for a review). Based on these findings and others, some have concluded that associations between purported environmental predictors and positive life outcomes are partly explained by intelligence (Gottfredson, 2004).

One question that the findings of intelligence researchers led to is whether a common factor underlies the concomitants of psychopathology or happiness. With respect to psychopathology, there is considerable evidence that personality traits are responsible for comorbidity (Kendler and Myers, 2010). Neuroticism is a particularly important risk factor for psychiatric disorders ranging from affective disorders, such as depression, to personality disorders, such as borderline personality disorder, substance abuse disorders, anxiety disorders, and eating disorders (Ellickson-Larew et al., 2013; Samuel et al., 2013; Hettema et al., 2004). Critically, Neuroticism and other personality dimensions appear to explain some overlap between disorders (Kendler et al., 2007; Khan et al., 2005). Similarly, Caspi et al. (2013) showed that high Neuroticism, low Agreeableness, and low Conscientiousness were associated with their p-factor.

Personality traits are also related to lower and higher SWB. Meta-analyses have shown that individuals who score higher on measures of SWB and related constructs tend to score lower on Neuroticism and higher on Extraversion, Openness to Experience, Agreeableness, and Conscientiousness (DeNeve and Cooper, 1998; Steel et al., 2008). Furthermore, a 40-year prospective found that the associations between Neuroticism and SWB were mediated via the former's impact on psychological distress and health problems (Gale et al., 2013). No such mediation effects were found for Extraversion. To summarize, personality accounts for just under half of the variance in SWB and these effects may reflect the impact of personality on other factors.

There is also evidence that the associations between personality dimensions and SWB

are not exclusive to humans. Studies of animal personality have shown that, along with species-specific dimensions, such as those related to social rank, variants of many human personality dimensions are present in other species (see Gosling, 2001 for a review). While assessing personality in animals seems farfetched, personality traits in nonhuman animals evidence many characteristics exhibited by human personality traits, including inter-rater agreement, heritability, stability over time and across situations, and associations with outcomes, including behavior, health, and longevity (see Gosling, 2001 for a review).

There is also evidence that something akin to happiness or SWB can be measured in nonhuman animals. Using a four-item measure, King and Landau (2003) found that human raters could reliably assess chimpanzees on four aspects of SWB: the balance of positive and negative moods, pleasure derived from social interactions, the ability to achieve goals, and global satisfaction. Principal components analysis indicated that a single dimension accounted for correlations among these items. Moreover, they found that this measure was stable over time and that around half of its variance could be accounted for by personality, with higher scores on the personality dimensions Dominance, Extraversion, and Conscientiousness (but not Agreeableness, Neuroticism, or Openness) being associated with higher SWB. These findings have been mostly replicated in chimpanzees in Japanese zoos (Weiss et al., 2009) and in other species, including orangutans (Weiss et al., 2006), rhesus macaques (Weiss et al., 2011b), and Scottish wildcats (Gartner and Weiss, 2013), all rated using the same or highly similar SWB scales.

Genetic determinants of covitality

The observation that personality and SWB correlations are consistent across species supports the notion that SWB is more than a reflection of a person's circumstances or demographic characteristics. This fact motivated researchers to try to understand the mechanisms

underlying these associations. Was covitality a result of unintended overlap in construct measurement, a trait-state dependency, or a common influence from some other source?

One hypothesis was that the genes underlying the covitality factor would be observable as genetic correlations between traits indicative of covitality. Several findings from the human literature led to this hypothesis. These include the findings that shared genetic effects explain the comorbidity between psychopathologies (Kendler et al., 2007; Middeldorp et al., 2005), and between high Neuroticism and depression (Kendler et al., 2006). As reviewed in this volume and elsewhere, SWB is heritable, with genetic contributions to variance estimated at ~40% based on twin studies (Bartels and Boomsma, 2009; Røysamb et al., 2002; see Chapters 5 and 10). Furthermore, across timespans of up to ten years, the stable variance in SWB has been attributed mostly to genetic causes (Nes et al., 2006; Lykken and Tellegen, 1996). Similarly, personality traits show heritabilities of around 50% (Bouchard and Loehlin, 2001), with stable influences largely reflecting genetic effects (Blonigen et al., 2008; Bratko and Butkovic, 2007; Viken et al., 1994). For SWB and personality, environmental effects are of the non-shared type (i.e., unique to the individual; see Chapter 3). In light of the similar (additive genetic and non-shared environmental) causes of variation in SWB and personality, it becomes reasonable to ask whether causes of variation overlap between these traits.

Determining the extent of common genetic and non-shared environmental effects on two or more traits and personality requires genetically informative designs. One such design is a multivariate extension of the classic twin design (see Chapter 3). In classical twin designs, the MZ and DZ cross-correlations, that is, the correlation of twin 1's score on one phenotype (e.g., SWB) and twin 2's score on a different phenotype (e.g., Extraversion), are compared instead with correlations on the same phenotype (Plomin, 1986). The difference between the MZ and DZ cross-correlations multiplied by two gives a rough estimate of the

genetic component of the phenotypic association of the two variables, termed the *bivariate heritability*. This estimate is equal to the multiplication of the square root of the heritability of each variable by the genetic correlation of the variables (i.e., their standardized genetic covariance). Note that environmental contributions to covariance and environmental correlations can similarly be derived. The expected cross-twin correlation between SWB in twin 1 and the personality trait in twin 2 is equal to h^2·(A correlation) + c^2·(C correlation) for MZs, and ½h^2·(A correlation) + c^2·(C correlation) for DZs (Finkel and McGue, 1993), where A refers to additive genetic variance and C to common (or shared) environmental variance. These equations will reduce if C effects are equal to zero, as expected for SWB and personality. Figure 9.1 depicts the path model of twin relationships for an additive genetic and non-shared environment model in the bivariate case.

<Insert Figure 9.1 here>

Another genetically informative design is that of a pedigree, where statistical separation of genetic and environmental components of variance is enabled by comparison of the phenotypic covariance between all pairs of relatives using information about their known genetic relations—termed an "animal model" (Lynch and Walsh, 1998). For example, the relatedness between parent–offspring pairs is .5, for grandparent–grandchild it is .25, and for first cousins it is .125. This information conditions the random additive genetic effect in the linear mixed model (typically used to derive estimates of genes and environment). Common environmental effects will be confounded with those of maternal and dominance effects unless these are also specified in the model. Sampling across generations and inclusion of half-siblings enables unique identification of these specific variance components (Wilson et al., 2010). In a univariate model specifying, for instance, genetic and maternal effects, an individual's trait value (y) is predicted by the equation, $\mathbf{X}\tau + \mathbf{Z}u + \mathbf{Z}m + e$, where vectors τ = fixed effects, u = fitted random additive genetic effects, m = fitted random maternal effects, e

= residual error effects, and **X** and **Z** are the respective associated design matrices for fixed effects, and random additive genetic and maternal effects. Bivariate modeling is an extension of the univariate case, in which the variance components of the observed phenotypic covariance between the two traits of interest is specified. For genetic mediation of the phenotypic correlation, the cross-trait cross-pedigree member correlation should be higher in pedigree members who are more closely genetically related. The animal model has mostly been used in nonhuman animal research, but because research on the genetic relationship between personality and SWB was originally carried out in nonhuman primates it is relevant to our exploration of the topic.

Weiss et al. (2002) were the first to examine the common genetic etiology of personality and SWB, though they did so in chimpanzees, and focused on Dominance, a trait that taps aspects of high extraversion and low neuroticism. They reasoned that, as chimpanzees and humans are genetically very similar (Chimpanzee Sequencing and Analysis Consortium, 2005), their results would yield clues as to the associations between human personality and SWB. Using the animal model, they estimated a fixed effect of zoo (common environment) and random effects, including additive genetic, maternal genetic, maternal (care-giver) environment, and non-shared environment (incorporating measurement error). Results showed heritabilities of 66% and 40% for Dominance and SWB, respectively, and respective non-shared environmental effects of 27% and 35%. While heritable maternal effects were present for SWB they were not present for Dominance, and thus did not contribute to Dominance and SWB covariance. Shared zoo effects were minimal. There was a perfect genetic correlation between Dominance and SWB, with much less overlap between the non-shared environmental factors influencing the traits (r_e = .11). The bivariate heritability of Dominance and SWB was 51%, with genes explaining about 93% of the covariance between both.

Based on this exciting finding, similar effects were investigated in humans. Using adult twin pairs from the MacArthur Foundation Survey of Midlife Development in the United States (MIDUS) (Kessler et al., 2004), the genetic and environmental contributions to covariance between the Big Five personality dimensions and SWB were estimated. With the exception of Openness, all traits showed evidence of dominant genetic rather than common environmental sources of variance; therefore, an additive genetic, dominant genetic, and non-shared environmental multivariate model was specified. There was insufficient power to support dominant genetic effects, with the resulting model capturing additive genetic and non-shared environmental effects such that a general additive genetic factor influenced personality and SWB. In addition to the effects shared between SWB and the general additive genetic factor, genetic effects were shared between SWB and (in order of importance) Neuroticism, Extraversion, and Conscientiousness. All genetic effects on SWB therefore overlapped with personality (in line with the original findings in chimpanzees) and the heightened genetic relationships with Neuroticism, Extraversion, and Conscientiousness mimicked their heightened phenotypic correlations reported in the literature (DeNeve and Cooper, 1998; Steel et al., 2008).

That genetic variation in SWB overlapped completely with genetic variation influencing measures of personality in both chimpanzee and humans prompted the question of whether these correlations arose independently in each of these highly social species. Adams et al. (2012) therefore investigated the genetic and environmental structure underlying orangutan SWB and personality, arguing that comparison of the structure in these semi-solitary species—closely related to humans and chimpanzees—could help dissociate ancestral versus derived adaptations. They found that orangutan personality and SWB were primarily influenced by non-additive genetic effects. In the multivariate model, although non-significant due to insufficient power, 15% of the additive genetic and 18% of non-additive

genetic variance in SWB overlapped with personality (Extraversion, Dominance, Neuroticism, Agreeableness, and Intellect). The degree of genetic overlap was much lower than that found in chimpanzees and humans. They concluded that whereas the association between personality and SWB is probably ancestral in great apes, the increased genetic overlap in humans and chimpanzees might have recently evolved. An alternate explanation was that personality and SWB in orangutans became more genetically uncoupled in their divergence as a species.

Although Weiss and colleagues (2002) did not explore the possibility of genetic non-additivity in chimpanzee SWB and a later study of a common factor defined by emotional well-being, psychological well-being, and social well-being by Keyes et al. (2014) did not explore this possibility in humans, evidence of genetic non-additivity for Neuroticism and life satisfaction overlap was found in a study of humans by Hahn and colleagues (2013). This latter finding is compatible with variation driven by similar mutation and selection processes (Adams et al., 2012), but whether this evolved independently or resulted from long-term selection common in the ancestors of humans and orangutans is not known.

Before considering possible explanations, it is important to understand that, while we interpret these findings as revealing biological pleiotropism, we cannot rule out the possibility that they reflect statistical pleiotropism (Carey, 1988). One explanation for the evolution of individual differences in these traits is balancing selection conditioned by environmental heterogeneity (Penke et al., 2007), although evidence for mutation–selection balance has also been found (Verweij et al., 2012). Which evolutionary theory can explain genetic variation in SWB? For example, can low mate preference values for SWB discount theories of mutation–selection balance as has been argued for personality? If so, does this imply that covitality is not central to general fitness but more a by-product of a fitter organism (see Chapter 4)?

Figueredo et al. (2004, 2007) examined the phenotypic and genetic correlations underlying traits in the MIDUS dataset. They found that self-reported SWB, positive affect, general health, negative affect (reversed), and medical symptoms (reversed) loaded onto a common factor. This factor was associated with two additional factors, one describing individuals who were emotionally stable, extraverted, open to experience, agreeable, and conscientious, and a second describing a reproductive life history strategy characterized by investment in the rearing of offspring, delay of gratification, and closer community and family ties. These phenotypic factor loadings and correlations were largely conserved when examining genetic correlations.

In a twin study, Steger et al. (2007) investigated the heritability of the 24 character strengths in the "Values in Action Inventory of Strengths" and their phenotypic and genetic correlations with the Well-being, Social Potency, Achievement, Social Closeness, Stress Reaction, Alienation, Aggression, Control, Harm Avoidance, Traditionalism, and Absorption scales of the Multidimensional Personality Questionnaire (Tellegen and Waller, 2008; Tellegen, 1982). They found that 20 character strengths were at least 30% heritable and that the environmental effects influencing these traits were mostly non-shared. They also showed that the genetic effects influencing character strengths were largely shared with personality. Finally, they found little evidence of overlap in non-shared environmental effects between character strengths and personality, much like the findings for SWB and personality.

Why do personality traits underlie positive traits?

How might one explain the genetic overlap among positive psychological characteristics and between those characteristics and normal personality variation? Before exploring this question one must rule out the possibility that SWB and other positive traits are nothing more than different measures of personality. One way to do so is to show that the independent non-

shared environmental effects detected in behavior genetic studies of covitality in humans (Hahn et al., 2013; Weiss et al., 2008; Steger et al., 2007; Keyes et al., 2014) and nonhuman primates (Weiss et al., 2002; Adams et al., 2012) do not reflect method variance. Doing so requires using multitrait–multimethod designs (Campbell and Fiske, 1959; Riemann and Kandler, 2010).

Although the hypothesis that SWB and other positive traits are nothing more than measures of personality has not been tested within a behavioral genetic framework, as no such studies have used multitrait–multimethod designs, there are three reasons to believe that common genetic causes do not reflect common method variance. First, the personality and SWB measures in the ape studies were derived from ratings by multiple raters (Weiss et al., 2006; King and Figueredo, 1997; King and Landau, 2003). As such, via aggregation (Weiss et al., 2002), or by partitioning covariance into rater- and animal-based effects in the animal model (Adams et al., 2012), these studies reduce the ability of common method variance to lead to correlations. This is highlighted by the fact that, among chimpanzees, SWB and Dominance items load onto distinct factors (Weiss et al., 2002) and that there is no evidence of a higher-order personality factor in chimpanzees or orangutans (Weiss et al., 2011a). Second, the genetic correlation between personality and SWB in orangutans held when adjusting for correlations arising because of common method (or rater) effects (Adams et al., 2012). Third, maternal effects influenced SWB but not Dominance in chimpanzees (Weiss et al., 2002).

If covitality is not merely an artifact owing to common measurement variance or underlying personality traits, what mechanisms might explain the overlap between positive traits and personality? Where positive psychological traits or latent constructs are shown to differ in their genetic and environmental structure, and given other model assumptions are satisfied, twin studies can be used to model the direction of causation between two traits

(Heath et al., 1993). Comparison of the patterns of cross-twin cross-trait correlations enables falsification of competing causal hypotheses. Such analyses, then, can suggest causality among associated positive traits or shared (pleiotropic) genetic effects.

Using SNP genotyping arrays, genetic pleiotropy (and potentially causality direction) can also be demonstrated. With this method phenotypic variation in one positive trait is predicted by a polygenic score that is calculated by forming a composite of the genetic effect sizes of SNP markers estimated via genome-wide association analysis of another positive trait. Arguably, as in cross-lagged panel designs (Cook and Campbell, 1979), direction of causation would be supported if the polygenic scores predicted phenotypic variation from one trait to the other, but not the converse,[4] given that the reliability of each of the measures and their genome-wide association results were the same.

An alternative design to tease apart mechanisms underlying covitality is to study MZ twins discordant for personality profiles related to SWB or other positive traits. One can then quantify the divergence of related positive traits between these twins, and ideally within a longitudinal framework. Whether all positive traits diverge in the same manner or some more than others might aid our conceptualization of covitality. Biographical data has been used in such a design to identify potential environmental factors that contribute to trait dissimilarity for depression (Kendler and Halberstadt, 2013).

Evolutionary hypotheses

There has been considerable speculation and work on the evolutionary mechanisms that maintain heritable variation in SWB and other positive traits (see Chapter 4 in this volume),

[4] Of course, as with cross-lagged panel designs, this approach would not completely rule out alternative causal explanations.

but these origins are unclear. We suggest that covitality offers insights and new hypotheses about these questions for SWB and other positive psychological traits.

One possibility is that positive psychological characteristics may be associated with one end of the life history strategy continuum. The concept of the life history strategy was developed within ecology and evolutionary biology (see Figueredo et al., 2006 for a discussion and review). Initially, species or populations were characterized as either *r*- or *K*-selected. Species or populations that are *r*-selected invested resources in mating and reproduction. Individual members of such species (e.g., rabbits) tend to be shorter-lived, reproduce sooner and more often, and develop more rapidly. Species or populations that are *K*-selected invest resources in the rearing of young and in development. As such, members of long-lived species (e.g., elephants) have fewer offspring, invest more heavily in their offspring, and develop more slowly.

The studies by Figueredo and his colleagues (2004, 2007) that support the hypothesis that life history strategies underlie this association could be criticized for relying strictly on self-reports. However, this hypothesis is also supported by findings showing that personality profiles related to SWB (DeNeve and Cooper, 1998; Steel et al., 2008) and other positive psychological characteristics (i.e., lower Neuroticism and higher Extraversion, Openness, Agreeableness, and Conscientiousness) predict longevity and health (Roberts et al., 2007; Chapman et al., 2011; Deary et al., 2010), and studies showing that genetic correlations underlie the links between SWB and actual illness (Røysamb et al., 2003).

The association between personality and positive psychological characteristics, and covitality more generally, may also be the products of sexual selection. In other words, positive psychological traits such as SWB may be hard to fake signals of good genetic quality, that is, a genome with low mutation load (see Penke et al., 2007 for a discussion), good parenting abilities, or both (Miller, 2001). The hypothesis that happiness is the human

or ape equivalent of a peacock's tail was proposed by Weiss et al. (2002) to explain the perfect genetic correlation between SWB and the Dominance domain in chimpanzees. The chimpanzee Dominance domain, although not synonymous with rank or social standing, is comprised of traits related to low neuroticism, high extraversion, and low agreeableness (King and Figueredo, 1997) and has thus been likened to "competitive prowess" (King and Weiss, 2011). Ethological descriptions of alpha male chimpanzees in the wild (Goodall, 1990; Van Lawick-Goodall, 1971) and in captivity (de Waal, 2000) as often swaggering, fearless tacticians that are able to recruit allies support this view. Additional evidence comes from studies in Barbary macaques, another nonhuman primate species, indicating that personality domains similar to Dominance predict concurrent and future rank (e.g., Konečná et al., 2012). Given that high-ranking males and females are higher in fitness as measured by reproductive success (Pusey et al., 1997; Wroblewski et al., 2009), SWB may signal rank and—for males who do not rear offspring—genetic quality.

So, is it possible that SWB and other positive traits offer a peek into the health of the organism and its suitability as a parent? Human evidence in favor of this hypothesis comes from the study of virtues. Miller (2007) argued that the sexual selection hypothesis is consistent with the heritability and the association between virtuous traits and personality, the preference for virtuous characteristics by human females, and the heritability of virtuous traits. However, in the case of SWB for humans (Nes et al., 2010; Hahn et al., 2013; Lykken and Tellegen, 1996; Bartels and Boomsma, 2009; Weiss et al., 2008) and for orangutans (Adams et al., 2012), the fact that a considerable amount of variance is due to non-additive genetic effects is inconsistent with these traits being sexually selected.

The future of covitality

Upon writing this chapter we asked ourselves: What lessons for positive psychology research

does covitality hold? We came up with two answers. First, the science of positive psychology needs to be linked to the science of personality. This is a strong statement, but researchers studying SWB and other measures of psychological well-being have long realized this fact. The second point is that covitality, studied at the level of the phenotype and genotype, is crucial for understanding the nature of positive psychological traits. In other words, what positive psychological constructs exist and how they are interrelated with each other and functioning and life outcomes will be as important to a scientific positive psychology as the discovery of general intelligence or the development of the Five-Factor Model of personality.

A research program following these guidelines could include developmental studies with questions such as to what extent do stages of moral reasoning (Kohlberg, 1974) or personal growth (Maslow, 1943) track personality development? Questions about the evolution of positive traits could also be addressed. For example, to what extent are positive psychological traits other than SWB possessed by nonhuman animals, including the great apes, and how did these traits evolve? Some researchers (Hare et al., 2012) proposed that "self-domestication" against aggression led to the dramatically different societies of chimpanzees and bonobos despite there being less than 1 million years of separation time (Prüfer et al., 2012). Could similar processes have led to some of our virtues (Allman, 1999)? A recent study showing craniofacial feminization (that may have been accompanied by decreased aggression) in more recent humans suggest that this line of enquiry could be promising (Cieri et al., 2014). In addition, modern molecular genetic techniques, including GWAS and GCTA, as well as physiological research of human personality and positive traits and their animal homologues, are needed to further understand the architecture of these traits and their association with personality (see Chapters 10 and 16). Finally, intervention studies seeking to enable individuals to flourish should examine whether interventions tailored to individuals' personalities are more effective.

All-in-all, then, there is reason to be positive, and even optimistic, about the promise that covitality holds for psychology research and thinking. At the very least it will help sharpen up what positive traits are and what they are not. At most, we hope it represents the rebirth of a scientific humanistic psychology.

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Figure 9.1 Path diagram showing the mathematical relations between MZ and DZ twins reared together across two phenotypes. Only additive genetic (A) and unique environment (including measurement error; E) factors are displayed.